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Acid Sensitive Ion Channels as Target of Hydrogen Sulfide in Rat Trigeminal Neurons

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Abstract

© 2016, Springer Science+Business Media New York. Hydrogen sulfide (H₂S), an endogenous gasotransmitter, was proposed to act as a signaling molecule in a wide variety of pathophysiological processes including inflammation and nociception. Acid-sensing ion channels (ASICs) are voltage-insensitive, proton-gated cation channels activated by extracellular acidosis, and are involved in various pathologies of the nervous system including ischemia, stroke, and migraine. In this study, the effect of the H₂S donor-sodium hydrogen sulfide (NaHS) on the firing of trigeminal (TG) nerve was explored using suction electrode recordings in peripheral branches of the TG nerve in isolated rat meninges, and patch clamp recordings of ASIC currents in isolated TG neurons. NaHS (100 μ M) increased the action potential frequency of TG nerve and bath application of NaHS increased the amplitude of ASIC currents triggered by focal application of low pH solution on isolated TG neurons. We propose that activation of ASICs by H₂S during chronic inflammation process contributes to the increased excitability of the TG system and may be implicated in the generation of nociceptive firing underlying migraine pain.

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Keywords

Acid-sensitive ion channels, Hydrogen sulfide, Pain, Trigeminal nerve